Epidemic Collapse

SIR,—Referring to the mysterious illness among pupils at a girls' school reported in the *B.M.J.* for 16 October (p. 950) the fact that laboratory investigations so far have proved negative prompts me to relate an incident with which I was concerned at a mixed secondary modern school some years ago.

I was called to this school one morning by the head teacher as a number of pupils had been taken ill. When I arrived I found that some of the children had already been sent home not feeling well. Others were lying in classrooms and corridors exhibiting various stages of "collapse." Among those I examined some had tremors, some were overbreathing, and many complained of headache, or pain in the chest and abdomen. Otherwise the results of clinical examination for any gross abnormality were negative. All recovered spontaneously sooner or later when left alone.

The curious fact was that none of the boys seemed to be affected, which lent support to the opinion I formed at the time that this incident was in the nature of a mass hysterical manifestation following the sight of the first casualty that morning (probably a normal fainting attack) during assembly.—I am, etc.,

Dover, Kent. C. F. LYNCH.

Hysteria and "Royal Free Disease"

SIR,—In reporting that psychiatrists consider the "mystery illness" among schoolgirls in Blackburn to be attributable to mass hysteria, Dr. Alfred Byrne, medical correspondent of the *Sunday Times*, remarks "Some of the nurses involved in the 'encephalitis' outbreak at the Royal Free Hospital 10 years ago ended up with this." The Medical Correspondent of the *Daily Telegraph* (18 October) makes a similar observation. These tend to convey the impression that hysteria was the predominant feature of that outbreak. While it is true that a "functional overlay" was a not uncommon feature in many cases, the problem of "Royal Free disease" can be brought into perspective only if the following facts are noted:

(1) The outbreak at the Royal Free Hospital was not an isolated incident. A large epidemic of the same disease occurred in Iceland in 1950, and permanent sequelae, both neurological and psychological, were recorded. Outbreaks were also reported from Adelaide (1951), Coventry (1954), New York State (1954), Durham (1955), and Athens (1958).

(2) During the summer of 1955, and throughout 1956, 1957, and 1958, many cases of the disease were treated in the infectious diseases department of the Royal Free Hospital at Hampstead. Indeed, the condition has been endemic in North London since 1955 (although it may be significant that no cases were seen during the very cold winter of 1962–3).

(3) Since October 1964 some 90 cases have been seen in a geographically circumscribed area in North London, and some of these have been severe enough to require hospital treatment.

(4) Many patients, both in 1955 and at the present time, show clinical evidence of severe involvement of the central nervous system with no hysterical overlay.

(5) Most cases have a low-grade pyrexia, cervical lymphadenopathy, scattered myalgic foci, and abnormal electromyograms and/or electroencephalograms. Dr. Ruth Harris and Dr. Jean Kennedy, of the Department of Electroencephalography at the Royal Free Hospital, have demonstrated abnormal encephalograms in 38 patients even after a considerable period from the onset of the illness.

In presenting a paper on the disease at a recent meeting of the World Union for Social and Prophylactic Medicine at Mittendorf, Austria, I was interested to find that a German physician, Dr. Frisch, of Freiburg, was thoroughly familiar with the condition and was able to show photographs illustrating severe neurological damage. It would be singularly unfortunate if the impression was created that this still prevalent and unexplained condition were thought to be the result of mass hysteria.—I am, etc.,

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"Winter Vomiting" and Collapse

SIR,—On 11 October our daughter aged 11 woke from sleep with severe vomiting and continued retching every twenty minutes for the next three hours. The following day she had a temperature of 99° F. (37.2° C.), slight anorexia, and dizziness. She returned to school and was well the remainder of the week until 17 October, when she complained of dizziness in the afternoon. She was well next morning but sent home from school with dizziness at midday. On 18 October she was slightly dizzy and remained at home. At 8.30 p.m. she complained of severe high epigastric pain going through to the back, and whilst I helped her upstairs she collapsed, was pulseless, sweating, and ashen. She remained unconscious possibly half a minute, her pulse was thready for the next two hours, and checked half-hourly remaining at a rate of 86. Her temperature was subnormal all night. She had acute high epigastric tenderness on palpation, no rebound, no guarding; rectal examination was negative, no nausea or vomiting. She slept fitfully with bouts of upper abdominal colic. By morning her temperature was 99° F. (37.2° C.) and pulse remained relatively slow at 84.

A consultant surgeon and paediatrician were called early morning and after discussion it was felt that this acute episode was possibly "winter vomiting." Her blood count was normal.

The epigastic tenderness persisted for several days and her general malaise gradually lessened over the next week. There was no nausea or vomiting during this time, or signs of meningeal irritation. The acute collapse presented as some abdominal emergency, but careful observation over a period of hours did not give clinical signs to substantiate this.

In retrospect, while conducting surgery on 7 October, I suddenly felt extremely ill, sweating, and collapse, and dizziness with nausea. Fortunately, I managed to telephone my partner and recall fumbling to replace the receiver, before transient loss of consciousness. I was taken home somewhat ashen in colour and recovered sufficiently in early evening to see my patients. I had Stemetil (prochlorperazine) to relieve the dizziness and nausea.

I trust that this record of personal observation may indicate the acute and dramatic onset which this syndrome may present.—I am, etc.,

Leamington Spa, Warwickshire. M. DOREEN BULL.

Treatment of Lung Cancer

SIR,—Drs. H. A. Hartley and B. S. Tate (2 October, p. 822) have pointed out that a patient may seek medical advice and the correct diagnosis may be made, but the treatment of an early cancer lesion may be delayed because of the delay at hospital level. This letter repeats what has already been described in another scientific journal, for cancer at a different site.[1]

It is possible that no efforts can be made to improve the treatment of the early lesion because of economic factors. It may be that the cure for cancer, which would add another 100,000 elderly citizens to a population already competing for housing, etc., could be a national economic disaster. If this is not the reason, then there is no excuse for treating patients with cancer inefficiently, and, as Drs. Hartley and Tate reported, to wait three months after diagnosis of cancer can at best be described as inefficient. No one could describe it as economic. Steps should be taken to investigate the causes of delay. How often is it due to inadequately trained personnel, how often to shortage of beds, how often to incomplete investigation?

The general public, who are not fools to be easily hoodwinked, are beginning to ask why is there delay in getting treatment when cancer propaganda preaches that early treatment may mean a cure?

The waiting in out-patients has been investigated by organization and methods teams, but at best this delay was a social inconvenience. The delay and misdiagnosis in the treatment of cancer may cost life. Both the patients and their doctors are well aware of hospital delay before treatment. Are those responsible for hospital policy equally aware of this?—I am, etc.,

London S.W.1. DAVID WALLACE.

REFERENCE

1 Wallace, D. M., and Harris, D. L., *Lancet*, 1965, 2, 332.

Sequelae of Thorotrast

SIR,—Your leading article (2 October, p. 771) and the discussion of the follow up of 2,377 patients by da Silva's group, in whom Thorotrast was administered between 1930 and 1952,[1] reminds one forcibly of the importance of a controlling body for the marketing of drugs.

In 1930–2 I carried out investigations[2–4] on a large number of rabbits and rats with the preparation Heyden 1073a (which was marketed later as Thorotrast). These investigations showed that Thorotrast was stored in the reticulo-endothelial cells mainly of the liver, spleen, bone-marrow, and brain-covering membranes; the organs, especially the liver, displayed fibrosis similar to atrophic cirrhosis; phagocytosis decreased 6 to 12 months after administration from the normal 38% to 4.2%; capillaries and small vessels